Research paper

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PII: S0008-8749(18)30076-5
DOI: https://doi.org/10.1016/j.cellimm.2018.02.013
Reference: YCIMM 3773

To appear in: *Cellular Immunology*

Received Date: 19 July 2017
Revised Date: 18 February 2018
Accepted Date: 20 February 2018

Please cite this article as: L. Wang, H.K. Wai Law, Immune complexes suppressed autophagy in glomerular endothelial cells, *Cellular Immunology* (2018), doi: https://doi.org/10.1016/j.cellimm.2018.02.013

Immune complexes suppressed autophagy in glomerular endothelial cells

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Abstract

Lupus nephritis is an immune-complexes mediated glomerulonephritis. Vascular lesions and endothelial cell injuries are common in lupus nephritis and important for renal damage. However, the precise mechanisms by which immune complexes lead to endothelial cell injuries are still unclear. Autophagy is a conserved metabolic process and shows protective roles in many cell types and diseases. In present study, we investigated whether immune complexes could affect autophagy and participate in endothelial dysfunctions. Heat-aggregated gamma globulin (HAGG) was used to substitute immune complexes. Glomerular endothelial cells (GECs) were incubated with HAGG and autophagy-related markers were evaluated. Results showed that HAGG suppressed autophagy in GECs, through Akt/mTOR-dependent pathway. The combination of HAGG and tumor necrosis factor-alpha suppressed autophagy in GECs and further decreased cell viabilities. The suppressed effects of HAGG on GECs autophagy and viability, especially under inflammatory microenvironment, may provide new views for explaining the mechanisms of renal impairments in lupus nephritis.

Keywords

Lupus nephritis; Glomerular endothelial cell; Heat-aggregated gamma globulin; Autophagy

Abbreviations

SLE, systemic lupus erythematosus; IC, immune complex; LN, lupus nephritis; ESRD, end-stage renal disease; GEC, glomerular endothelial cell; HAGG, heat-aggregated gamma globulin; 3MA, 3-methyladenine; LC3 (or MAP1LC3), microtubule-associated proteins 1A/1B light chain 3; p62 (or SQSTM1),